COMPARATIVE STUDY OF MAGNESIUM SULPHATE REGIMENS - PRITCHARD REGIMEN AND DHAKA REGIMEN IN THE MANAGEMENT OF ANTEPARTUM ECLAMPSIA

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CERTIFICATE

This is to certify that the dissertation entitled ***"COMPARATIVE STUDY OF MAGNESIUM SULPHATE REGIMENS – PRITCHARD REGIMEN AND DHAKA REGIMEN IN THE MANAGEMENT OF ANTEPARTUM ECLAMPSIA""*** *submitted by* ***Dr. G. Manjari*** *to the Faculty of Obstetrics and Gynaecology, The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch II (Obstetrics and Gynaecology) is a bonafide research work carried out by her under our direct supervision and guidance.*

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CONTENTS

INTRODUCTION

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Eclampsia is a form of Hypertensive encephalopathy with generalised convulsions associated with signs of preeclampsia during pregnancy, labour (or) within 7 days of delivery and not caused by epilepsy (or) other convulsive disorders. It is one of the important causes of mortality and morbidity during pregnancy, child birth & puerperium.

Of the estimated 7,00,000 maternal deaths every year world wide 10% to 15% are associated with Hypertensive disorder of pregnancy.

The incidence of Eclampsia in developing countries is 0.5 – 2% but 4.9%/10,000 in United Kingdom[1] and 1 in 2000 in Europe and developed countries.

Eclampsia accounts for 50,000 maternal deaths a year world wide.

The maternal case fatality rate is 1.8% and 35% of eclamptics will have one major complication.

Perinatal mortality rate in developed countries is less than 10/1000 births to as high as 80 (or) more/1000 births in developing countries. The

Collaborative Eclampsia Trial group found that the incidence of perinatal mortality in eclampsia ranges from 224 to 307/1000 cases of Eclampsia.[2] According to Mudhaliar, perinatal mortality in Eclampsia is 300 – 600/1000 cases of Eclampsia. The overall PNMR Eclampsia is 363/1000 cases of Eclampsia.

Although it is a standard practice to use anti convulsants for management of Eclampsia, the choice of agent is controversial. Until recently, the pharmacological treatment of eclampsia has been determined by geography, habit and tradition. Magnesium sulphate is the drug of choice in US, in Britain and in many parts of world.

Collaborative Eclampsia Trial shows – Not only does magnesium sulphate diminish the risk of further convulsions, but it also produces less maternal and neonatal morbidity than the other agents.[3]

A smaller study carried out at Dhaka Medical College at the same time as the CET came to exactly the same conclusions. The main difference between these 2 studies was the dosage regimen of magnesium sulphate.

The loading does of Dhaka was significantly less than that used by Collaborative Eclampsia Trial - 10g loading does compared with 14gm. The

lower dosage had been chosen because of small size of Bangladesh woman. The evidence was considered by a working party of Eclampsia in October 1997. It was agreed that the Regime validated for local use would be recommended for National use.[4] Guidelines were prepared and disseminated. With this regimen the mortality rates in Dhaka medical college have fallen dramatically.[5]

This study compares the Pritchard Regimen with Dhaka regimen of magnesium sulphate in management of Antepartum eclampsia.

AIM OF THE STUDY

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1) To study the effect of low dose Magnesium sulphate Regimen - Dhaka Regimen in Antepartum Eclampsia.

2) To compare the effects of Magnesium sulphate regimens – Dhaka regimen with Pritchard regimen.

    i) The efficacy of controlling convulsions

    ii) In preventing recurrent convulsions.

    iii) The incidence of complications.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Definition:-

Preeclampsia complicated by generalised tonic-clonic convulsions is termed eclampsia. Fatal coma without convulsions also called as eclampsia, however it is better to limit the diagnosis to woman with convulsions and to regard deaths in non convulsive cases as due to severe preeclampsia.

Depending on whether convulsions appear before, during (or) after labour, eclampsia is designated as Antepartum, Intrapartum (or) Post partum.

Etiology:

Etiology of Pregnancy induced Hypertension.

1) Abnormal trophoblastic Invasion:

In pre eclampsia, there is incomplete trophoblastic invasion of uterine spiral arteries[6]. Endothelial damage, insudation of plasma constituents into vessel wall, proliferation of myointimal cells & medial necrosis are observed.[7]

Lipid laden myointimal cells and macrophages – atherosis[8] causes obstruction of spiral arterioles.

2) Immunological Factors:-

The risk of preeclampsia is enhanced where formation of Blocking Antibodies to placental antigenic sites might be impaired. They arise where effective immunisation by previous pregnancy is lacking as in first pregnancy (or) in multiple pregnancy where number of Antigenic sites provided by the placenta is great compared to the amount of antibody – Beer (1978) [9]

Bardeguez and associates (1991)[10] – woman who develop preeclampsia have lower proportion of helper T cells (Th_1) than normotensive woman. Th_1/Th_2 imbalance with Th_2 dominance may be mediated by adenosine, which is higher in serum of preeclampsia woman than normotensives.

Preeclampsia common in woman with anticardiolipin antibodies, Antibodies associated with β_2 glycoprotein 1 appear more relevant. Immune complexes and anti endothelial cell antibodies may also be involved (Taylor and Roberts, 1999)[11]

3) Genetic Factors:

Ness (2003)[12] suggested that the tendency for preeclampsia is inherited. Cooper and Liston (1979) suggested that susceptibility to pregnancy induced Hypertension is due to single recessive gene[13] Chesley and Cooper

(1986) suggested single gene model[14]. Trogstad and coworkers (2004) suggested polygenic inheritance.[15] Kilpatrick and associates (1989) showed association between HLA-DR4 and proteinuric Hypertension.[16]

Ward and Zhang (2003) reported that woman heterozygous for angiotensinogen gene variant T_{235} had a higher incidence of preeclampsia of fetal growth restriction[17]. Morgan and colleagues (1995, 1999) did not confirm this, but they found that woman homozygous for the mutation had abnormal trophoblastic invasion.[18]

Polymorphisms of genes for TNF, Lymphotoxin –α and interleukin – 1β have been studied with varying results[19] (Hefler; Lachmeijer; Livingstor 2001).

Dizon Townsend and colleagues (1996) found higher incidence of factor V leiden mutations in pregnancy induced hypertensive woman. [20]

4) Nutritional Factors:-

A number of dietary deficiencies (or) excess have been blamed as a cause of eclampsia. Dietary taboos included are meat, protein, purines, fat, dairy products salt and others.

John and coworkers (2002) showed that a diet rich in fruits and vegetables have antioxidant activity with decreased blood pressure.

Zhang and associates showed that the incidence of preeclampsia was doubled in woman whose daily intake of ascorbic acid was less than 85mg.[21]

Carroli and colleagues (1994) implicated that calcium supplementation reduces the risk of preeclampsia.[22]

Belizan (1991), Lopez-Jeramillo (1989), Sanchez-Ramos (1994)[23] and their associates reported that mid pregnancy daily dietary supplementation of 2gm calcium significantly reduced the incidence of Hypertension.

5) Vasculopathy and the inflammatory Changes:-

In response to placental factors released by ischemic changes, (or) any other inciting cause, a cascade of events is set in motion (Redman and Sargent 2003)[24]

Staff and colleagues 1999 – suggested that the decidua also contains an abundance of cells when activated, can release noxious agents.[25] They serve as mediators to provoke endothelial cell injury.

Fass and colleagues 2000 and Gervasi 2001, showed that preeclampsia is due to an extreme state of activated leucocytes in maternal circulation.[26]

TNF –α and their interleukins may contribute to the Oxidative stress associated with preeclampsia.

Oxidative stress produce highly toxic radicals and injure endothelial cells (Manten and associates 2005)[27] modify their nitric oxide production and interfere with prostaglandin balance.

PATHOGENESIS

1) Vasospasm:

The concept of vasospasm was advanced by Volhard (1918)[28] based on direct observation of small vessels in nail beds, ocular fundi and bulbar conjunctiva. Wang and colleagues (2002) demonstrated disruption of endothelial junctional proteins.[29] Suzuki and co workers (2003) demonstrated ultrastructural changes in sub endothelial region of resistance arteries in preeclamptic woman.[30] Vasospasm may be worse in HELLP syndrome – Fischer and colleagues, 2000.[31]

2) Endothelial cell activation:-

Hayman, Roberts, Walker 2000 showed that clinical syndrome of preeclampsia result from widespread endothelial cell changes.[32]

(i) Increased Pressor responses:-

Abdul – Karim and Assali 1966 showed that normal pregnant woman develop refractoriness to infused Vasopressors.[33]

Woman with early preeclampsia, have increased vascular reactivity to infused nor epinephrine and angiotensin II.[34] (Raab and co workers 1956. Talledo and associates, 1968)

ii) Prostaglandins:-

Taylor and Roberts (1999) showed that endothelial prostacyclin production is decreased in preeclampsia that is mediated by phospholipase A_2.[35]

Platelet produced thromboxane A_2 increased and the prostacyclin : thromboxane A_2 ratio decreases. That favours increased sensitivity to angiotensin II that ends in Vaso constriction.

Chavarria (2003) given the evidence that these changes are apparent as early as 22weeks in woman who later develop preeclampsia.

iii) Nitric Oxide:

A potent vasodilator synthesized from L-arginine by endothelial cells. It is the compound that maintains normal low pressure vasodilator state characteristic of Feto placental perfusion – Myatt and co workers 1992.[36]

Wang and colleagues showed that preeclampsia is associated with decreased endothelial nitric oxide synthase expression, which increases cell permeability. It's production increased as compensatory mechanism in severe preeclampsia[37]. So increased serum concentration of nitric oxide is likely the result of hypertension, not the cause – Morris and colleagues 1996. [38]

iv) Endothelins:

Mastrogiannis and coworkers showed that these 21 amino acid peptides are potent vaso constrictors, and endothelin -1 (ET-I) is primary isoform produced by human endothelium.[39]

Taylor and Roberts (1999) showed that the placenta is not the source of increased ET-I.[40]

Sagsoz and Kucukozkan 2003 observed that treatment of preeclamptic woman with magnesium sulphate lowers ET-I concentrations.[41]

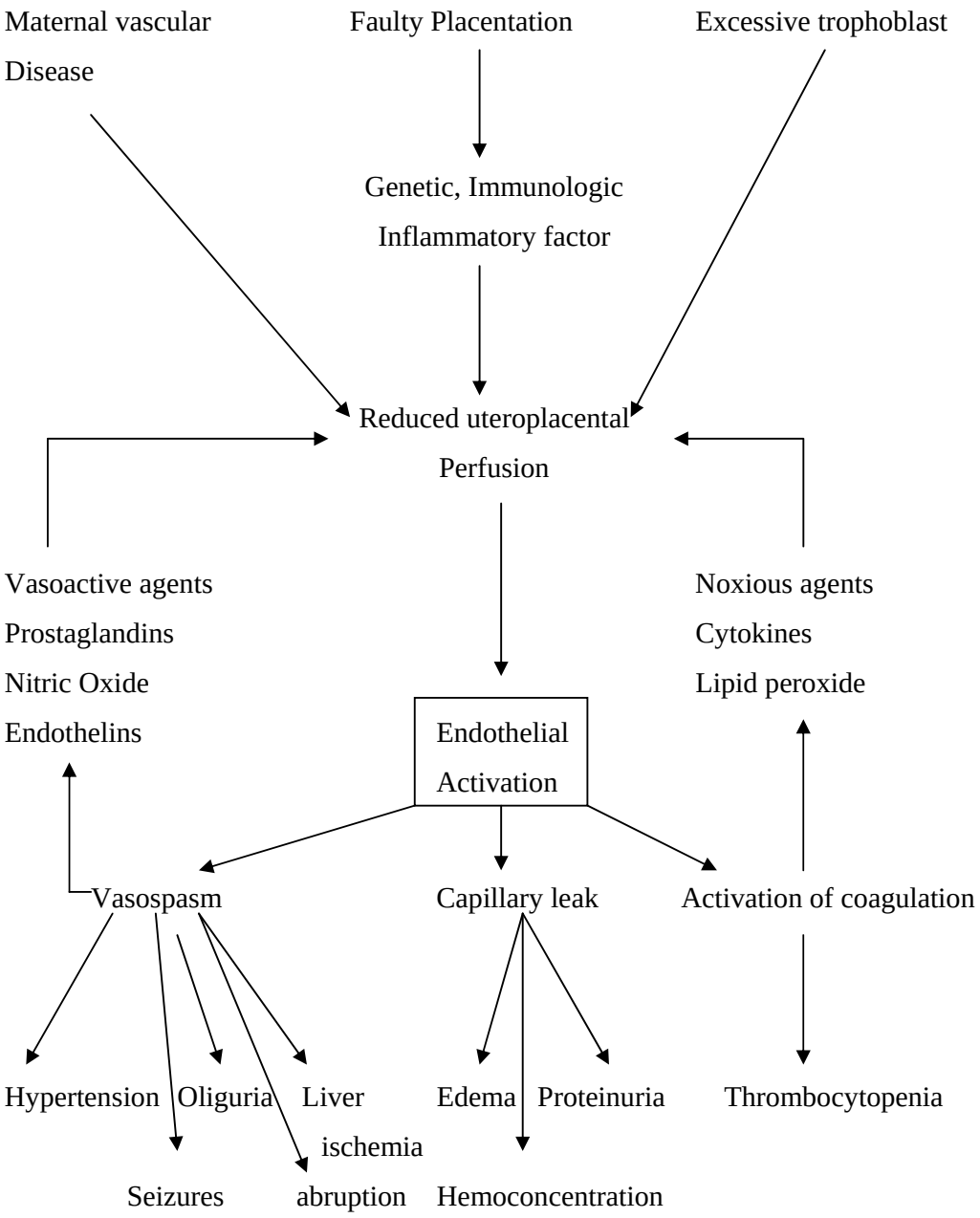
v) Angiogenic Factors:

Vascular endothelial growth factor (VEGF) and placental growth factor (PIGF), the glycosylated Glycoproteins are selectively mitogenic to endothelial cells.

Simmons and co workers 2000 showed the VEGP is increased in serum from woman with preeclampsia but the bioavailability is decreased[42].

In preeclampsia, the gene for Soluble Fms-Like tynosine kinase 1 (SFLt) is upregulated – Maynard & associates.[43]

Pathophysiology



Clinical Course of eclampsia

Eclampsia is most common in last trimester and becomes more frequent as term approaches.

Maternal hypoxia and lactic acidosis caused by convulsions, the fetus develops bradycardia, pulmonary edema may occur. Sudden death due to massive cerebral hemorrhage can occur. 10% of cases had blindness due to retinal detachment (or) occipital lobe ischemia and edema. Rarely eclampsia is followed by psychosis.

Differential diagnosis:-

Epilepsy, encephalitis, meningitis cerebral tumor, cysticercosis and ruptured cerebral aneurysm.

Complications of Eclampsia:

Placental abruptions (10%), Neurological deficits (7%) Aspiration pneumonia (7%) pulmonary edema (5%) cardiopulmonary arrest (4%) Acute renal failure (4%) maternal death (1%).

MANAGEMENT

I. General Management

It plays an important role in the management of eclampsia. The patient is nursed in a quiet room with a medical or nursing attendant always present. Pulse rate, respiration, blood pressure, colour, restlessness, urine output must be constantly observed. A mouth gag, airway and O_2 must be available. Patient is put in left lateral position in a railed cot. Throat is cleared of secretions and vomitus by intermittent suction. A soft firm mouth gag introduced in time will save injury to the tongue. An indwelling catheter in the bladder will give an accurate assessment of the urine output and will also prevent restlessness due to a full bladder (Dewhurst 1984). Blood pressure is measured half hourly till it is controlled and then second hourly. A record of grade of consciousness is maintained. Nutrition and hydration are maintained parenterally.

II. Anticonvulsant line of Management

History

In the early years of the 20^{th} century early intervention to achieve delivery was widely practiced. The mortality was very high. Later in 1930, Strongonoff introduced his conservative regimen of heavy sedation with morphia by injection, and chloral or bromide per rectum gave better results. Once the convulsions were fully controlled the membrane were ruptured.

In 1920-30 stomach and colonic lavage with magnesium sulphate was used to clear the patient of toxins and sedation with chloroform.

In 1951-1960 barbiturates were used. 'O' Donnel Browned used continuous thiopentone in 20% dextrose, but causes marked cerebral depression.

In 1961, lytic cocktail regimen using pethidine, chlorpromazine and promethazine was introduced by Dr. M.K.K. Menon [44]. He treated 1448 cases. Recurrence of fits was 15%. Maternal mortality was 2.4%.

Nager et al (1988) treated 98 cases with lytic cocktail (LC) with maternal mortality rate of 8.2%.

Llewellyn Jones (1961) conducted a study on 150 cases of eclampsia with lytilcocktail and the maternal mortality was 6.6% [45].

Lopez Liera used lyticocktail on 120 eclmaptic women and the maternal mortality was 11.7%.[46]

Lean et al (1968) have reported excellent results with large doses of chlordiazepoxide or diazepam.

Magnesium sulphate was first used by Horn in Germany in 1906 intra thecally [47]. In 1926 intravenous regimen of magnesium sulphate was reported by Lazard in Los Angeles and intramuscular regimen by Dorsett in St. Louis. However the popular intra muscular regimen of Pritchard was introduced in 1955 and intravenous regimen by Zuspan in 1964.

Phenytoin Sodium was introduced in management of eclampsia in 1987.

DOSAGE SCHEDULE OF VARIOUS REGIMENS

1. Menon's Regimen (1961)

0 Hours

25 mg chlorpromazine and 100 mg pethidine in 20 ml of 5% glucose given intravenously. 50 mg chlorpromazine and 25 mg promethazine given intramuscularly.

A drip of 20% dextrose containing 200 mg pethidine is set up and run slowly at a rate of 20-30 drops / minute.

0-4 hrs	Promethazine	25 mg Im
0-8 hrs	Chlorpromazine	50 mg Im
0-12 hrs	Promethazine	25 mg Im
0-16 hrs	Chlorpromazine	50 mg Im
0-20 hrs	Promethazine	25 mg Im
0-24 hrs	Chlorpromazine	50 mg Im
0-28 hrs	Promethazine	25 mg Im
0-32 hrs	Chlorpromazine	50 mg Im
0-36 hrs	Promethazine	25 mg Im
0-40 hrs	Chlorpromazine	50 mg Im
0-44 hrs	Promethazine	25 mg Im
0-48 hrs	Chlorpromazine	50 mg Im

Menon (1961) used lyticocktail in 1448 eclamptic women and maternal mortality was 2.2%.[48]

Llewllyn Jones (1961) in his study using lyticcocktail had a maternal mortality of 6.6%.[49] Lopez Liera (1982) in his study had a maternal mortality of 11.7%.[50] Bhalla et al (1994) in his study using lyticcocktail had a maternal mortality of 4.4%.[51]

2. Diazepam (1968)

A loading dose of 10 mg diazepam intravenously over 2 minutes followed by an intravenous infusion of 40 mg in 500 ml normal saline for 24 hours. Rate of infusion titrated against level of consciousness with the aim of keeping the woman sedated but arousable. Diazepam can cause respiratory depression. It is poorly excreted by the neonate which tends to be sedated, hypothermic and unable to breast feed for several days.

3. Chlordiazepoxide (1968)

An initial intravenous injection of 10 mg followed by a continuous injection of 100 mg in 500 ml of 5% dextrose at a rate of 30 drops / minute or titrated according to the sedation needed.

4. Clonazepam (1977)

1 mg intra venous bolus followed by an intravenous infusion of 2.5 mg in 500 ml of 5% dextrose.

5. Chlormethiazole (Duffs et al 1968)

It is an effective anticonvulsant. Intra venous infusion of 0.8% chlormethiazole in 500 ml of 5% dextrose at a rate of 2-4 gm / hour for first 5-10 minutes and 0.5-1 gm / hour thereafter to produce easily

arousable sleep. It has a short half life of 45 minutes, so does not produce prolonged sedation in the mother or neonate.

6. Magnesium Sulphate ($MgSo_4$)

In 1955 Pritchard initiated a standardized treatment regimen at Parkland Hospital.

In 1964 Zuspan initiated the intravenous magnesium sulphate regimen.

a) Pritchard Regimen

Loading Dose	Maintenance Dose
4 g of 20% $MgSo_4$ IV at a rate not exceeding 1 g / min	Every 4 hrs 5 gm of 50% $MgSo_4$ as IM on alternate buttocks after assuring
10 g of 50% $MgSo_4$ deep IM in buttocks	a) Patellar reflex is present b) Respiration are not depressed > 16 / minute c) Urine output > 100 ml in preceeding 4 hours
If convulsions persists after 15 minutes, give 2 g of 20% $MgSo_4$ at a rate not exceeding 1 gm / minute	$MgSo_4$ discontinued 24 hours after delivery

b) Zuspan's Regimen (1964)

Loading Dose	Maintenance Dose
4 g of 20% $MgSo_4$ IV at a rate not exceeding 1 g / min	1-2 g / hour by controlled infusion pump for 24 hours after delivery (concentration not to exceed 20%)

c) University of Tennessee guidelines for intravenous $mgso_4$

Loading Dose

Give 30ml of 20%$mgso_4$ solution (6g) in 100ml of 5% dextrose over 10 to 15minutes.

Maintenance Dose:

Add 20g of mgso4 (Four 10ml ampoules of 50% solution to 1000ml of 5% Dextrose and give intravenously at a rate of 100ml/hour (2g/hour). Adjust the rate of infusion to keep serum magnesium levels between 4.8 & 9.6mg/dl.

If serum magnesium levels are not available the dose is adjusted according to the patellar reflex and urine output in previous 4hour period.

Monitoring of Magnesium Toxicity:

- ★ Urine output atleast 30ml / hour.
- ★ Deep tendon reflexes should be present.
- ★ Respiratory rate should exceed 14/min.

d) Dhaka Regimen:-

This prospective study was undertaken at Dhaka Medical College Hospital between 25 March and 15 June 1998.

It has the approval of the Hospital research Ethics committee. The agreed guidelines for management of eclampsia patients were followed.[52]The regime used was that from the Dhaka study.[53]

4gm magnesium sulphate given intravenously slowly over 15 minutes along with 3gm given intramuscularly in each buttock as a loading dose-maintenance therapy was 2.5gm every 4 hours, given intramuscularly in alternate buttocks until 24 hours after administration of the first dose.

Patients were monitored every 30 minutes for the first 6hours, then hourly by observing respiratory rate, knee jerk, urine out put.

Phenytoin Regimen :-

Slater et al – Woman weight 40-50 kg	= 750 mg
51-70 kg	= 1000 mg
> 70 kg	= 1250 mg

First 250mg is diluted in 250m I of normal saline and infused a rate of 25mg/minute Rest given over 15minutes. After 12hours 500mg of phenytoin

given as intravenous infusion (or) orally.

ECTG(1995)[54]

- 1 gm by slow intravenous infusion over 20mts is the loading dose
- 100mg every 6th hourly for next 24hours

Dommisse(1990)[54]

- 0.5-1 gm intravenously over 20minutes
- 0.5gm over next 1 hour then
- 0.5gm over 4hours, 12hours over staring treatment.

Lucas et al(1995)[55]

- 1 gm intravenously over 1 hour is the loading dose
- 500mg orally 10hour later
- continued for 24hours post partum

Appleton[54]

- 10mg/Kg as intravenous infusion at a rate of 25-40mg/minute is the loading dose.
- 5mg/kg as intravenous infusion 5hours later.
- 200mg every 8hours initiated .12hours after second dose until 24hours post delivery.

Coyagi et al (1994) [56]

Used single dose of 900mg phenytoin sodium as intravenous infusion. Control of convulsions was adequate without need for any complicated drug related patient monitoring.

PHARMACOKINETICS

Magnesium Sulphate

It has a molecular weight of 246. 1 gm of magnesium sulphate has 98mg of elemental magnesium.

Distribution and plasma levels[57,58]

Infused magnesium sulphate is distributed rapidly throughout the entire extra cellular fluid space and some is taken up by bone but none by red blood cells. Intravenous loading dose of 4-6gm results in immediate plasma concentration of 5-9mg/dl and it falls to 3-4gm/dl in 60minutes. Within 90minutes 50% of infused magnesium moves into bones and other cells. By 4hours 50% of infused Magnesium is excreted in the urine.

Excretion

Magnesium is excreted almost solely by the Kidneys. 50% of the infused dose is excreted after 4 hours in urine. 99% of the bolus intravenous dose is excreted within 24hours.

Mechanism of action

Some believe its action to be mainly peripheral at the neuro muscular junction with minimal central effects. While some believe that the main action is central. Calcium entry into neurons is regulated by specific excitatory amino acid linked channels. Excitatory amino acids such as L-glutamate and L-aspartate are the major neuro transmitters in mammalian central nervous system. These neurotransmitters produce their effects by interacting with certain receptors on the cell surface, the excitatory amino acid receptors, N-methyl D-aspartate (NMDA) is the best characterised excitatory amino acid receptor sub type. NMDA receptor has its channel blocked by magnesium ion and thus blocking neuronal calcium influx. Thus magnesium has a central nervous system effect in blocking the seizure. Cotton and associates (1992) have shown that hippocampal seizures could be blocked by magnesium [59]

Magnesium sulphate is a potent vasodilator especially in cerebral vasculature thus relieving cerebral vasospasm which is thought to be a cause for eclampsia.

Other Actions

- Vaso dilatation in Vascular beds
- Increased uterine blood flow (Harbert &collegues)
- Increased renal blood flow
- Increased prostocyclin release by endothelial cells (Watson and Colleagues)
- Decreased plasma renin activity
- Decreased angiotensin converting enzyme levels
- Attentuation of vascular response to pressor substances
- Bronchodilatation
- Reduced platelet aggregation

Pharamacological effects

- Anti convulsant action
- Transient hypotensive effect
- Transient but mild decrease in frequency of uterine contractions but no change in the intensity of contractions.
- Clinically insignificant decrease in short term variability of fetal heart rate.
- No change in long term variability of fetal heart rate or fetal heart rate accelerations

Side Effects

First sign of magnesium toxicity is usually the loss of patellar reflexes that occurs usually at about 9-12mg/dl because of curariform action. So maintenance does of $MgS0_4$ is not to be given in the absence of patellar reflexes.

Early signs and symptoms of magnesium toxicity include nausea, feeling of warmth, flushing, somnolence, double vision, slurred speech and weakness. These symptoms usually develop at plasma levels of 9 to12mg/dl.

Muscle paralysis and respiratory arrest develop at plasma level of 15-17mg/dl. Hence respiratory rate is monitored closely.

Cardiac arrest develops at level a of 30-35mg/dl. Thus it is important to keep an ampoule containing 1 gram of calcium gluconate at the bedside for intravenous administration as an antidote in case of magnesium toxicity.

Tracheal intubation and mechanical ventilation for severe respiratory depression (or) arrest. There is a transient decrease in uterine activity during intravenous injection alone. It can cause a transient decrease in fetal heart rate variability, neonatal neuro muscular and respiratory depression hyporeflexia and low apgar scores. These effects were reported in preterm infants in

association with fetal growth retardation. In addition several recent studies reported no ill effects in term infants. It can cause excessive blood loss after delivery.

Efficacy and Safety

Rapidly effective, reliable & predictable duration of action, wide safety margin, non depressive and non toxic to the mother and baby, simple to administer and monitor in the clinical setting and readily available antidote. Serum magnesium can be measured to ensure therapeutic concentration but many practitioners are happy to omit biochemical monitoring because of its wide margin of safety.[60]

Duley et al (1995) in his study used clinical evaluation alone and showed that the there is no need to check serum magnesium levels Estimation of magnesium levels are useful in the management of treatment failures.

Drug interactions with Magnesium Sulphate

Agent	Effect	Recommendation
Depolarising / non depolarizing neuro muscular blockers	Increased activity of these agents	May need dosage reduction of neuro muscular blocking agents

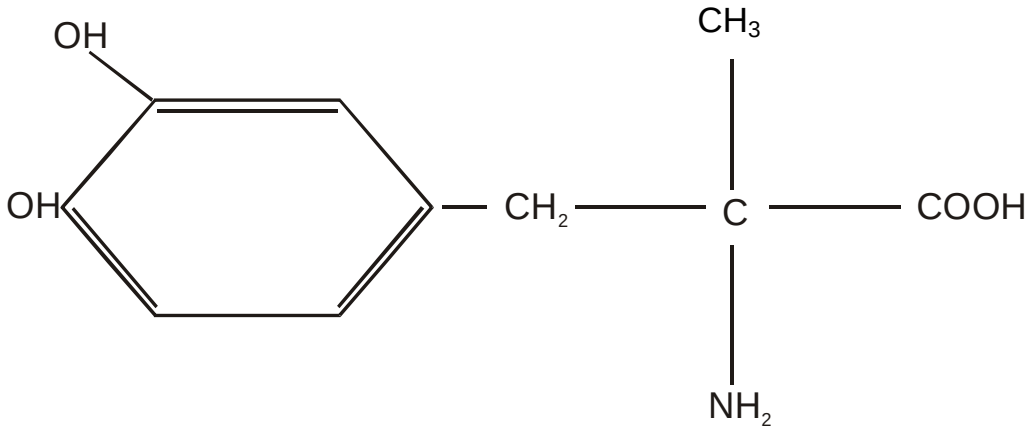
Agent	Effect	Recommendation
CNS depressants eg opioids, barbiturates, general anaesthetics	Additive CNS depression	May require dose reduction of CNS depressants
Nifedipine	Hypotension	Administer with caution and adjust nifedipine dosage if necessary

At the neuromuscular junction magnesium decreases the presynaptic release of acetylcholine and reduces the sensitivity of the post junctional membrane (motor end plate). Ghoneim and long reported that the action of succinylcholine (non depolarizing agent) are potentiated by magnesium sulphate. A single dose of succinylcholine can be safely used to facilitate tracheal intubation but may not apply when repeated dose of succinyl choline are used.[61]

When a patient is simultaneously exposed to magnesium sulphate and nifedipine some interaction might be expected as both are calcium channel blockers. Fenakel et al (1998) studied nifedipine in women who are receiving magnesium sulphate and found effective blood pressure control in 96% women without hypertension. It would appear that while a theoretical risk of interaction could exist in practice this is relatively uncommon.[61]

Alpha Methyl dopa

This was introduced to treat hypertension in 1960 by sjoerdesma



Pharmacological action

After oral (or) intravenous administration the hypotensive effect appears after 3 to 6 hours and 1-2 hours respectively. Hypotensive effect is associated with reduced cardiac output and total peripheral resistance. It does not reduce renal blood flow.

Mechanism of action

1. Inhibits adrenergic receptors in the vasomotor center
2. Inhibits renin release by the kidneys

Absorption and Excretion

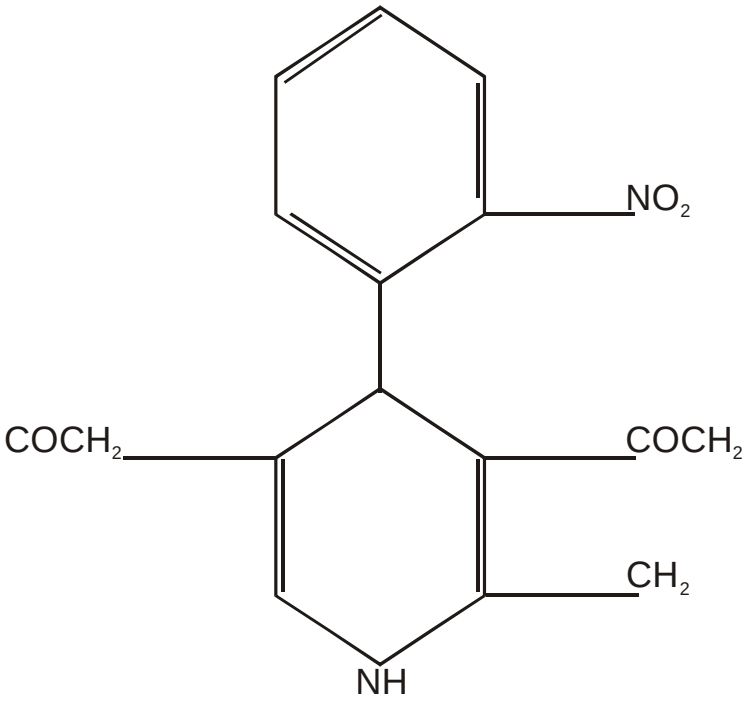
It is well absorbed orally and completely excreted in urine in 12 hours.

Adverse Effects

It commonly produces sedation, headache and fatigue. Other side effects are diminished Intellectual drive, drowsiness, forgetfullness, night 'mares parkinsonism, lactation and depression. Tolerance to antihypertensive effect is common. It can cause drug fever, altered liver function test (or) cholestatic jaundice. Rarely agranulocytosis, thrombocytopenia, gastrointestinal tract upset, constipation and skin rashes.

Nifedipine

It a calcium channel blocker, introduced in clinical practice in 1970 by Flerk, and Entus in 1971.



PHARMACOLOGICAL ACTIONS

It blocks the calcium transport through voltage dependent channel and thus inhibits the entry of extra cellular calcium ion necessary for the excitation contraction coupling in both the skeletal muscle and smooth muscle. It has a negative inotropic action on heart, relaxes the vascular smooth muscle in systemic as well as pulmonary circulation. Thus decreases the vascular resistance and the blood pressure. If causes a reflex tachycardia. It can be used orally or sublingually.

Side effects

It produces headache, tachycardia, dizziness, fatigue, orthostatic hypotension, leg cramps and skinrashes.

Interactions

Some have advised caution while using nifedipine with magnesium sulphate as both are calcium channel blockers, which may have a depressive effect on blood pressure.

Fenakel et al (1998) studied nifedipine in women who were receiving magnesium sulphate and found effective blood pressure control in 96% women without undesirable side effects and no cases of hypotension. It would

therefore appear that while a theoretical risk of interaction does exist. In practice this is uncommon.

Bhalla et al (1994) in his study used nifedipine and magnesium sulphate and had a good control of blood pressure.

Other Antihypertensives

Intravenous hydrallizine (or) intravenous labetalol produces remarkably effective control of the high blood pressure.

OBSTETRIC MANAGEMENT

After stabilizing the patient a detailed obstetric examination was made. If she is in labour, amniotomy was done and oxytocin infusion was started. If she is not in labour induction with prostaglandin E_2 gel done for a favourable cervical score.

Cesarean section was for obstetric indication, for failure of medical treatment.

I Stage

During labour fetal heart rate was ausculated every fifteen minutes, blood pressure was recorded every 2nd hourly. Intake, output was maintained strictly. Maternal pulse was recorded hourly. Cardiovascular system and

respiratory system were ausculated every 2nd hourly. Intravenous fluids preferably ringer lactate was infused at a rate of 1 ml/kg / hour.

II Stage

Completed by forceps, if indicated.

III Stage

Prophylactic methyl ergometrine was usually avoided to prevent the rise in blood pressure.

Blood Pressure was recorded every 2nd hourly until controlled and then' every 4th hourly for 48hours. All other vital signs and urine output were recorded every second hourly for 48 hours after delivery.

MATERIALS & METHODS

MATERIALS & METHODS

This study was conducted in Government Rajaji Hospital, Madurai during the period of July 2005 – July 2006.

60 consecutive patients with antepartum eclampsia were included in the study. Magnesium sulphate was used in control of convulsions. 30 patients were put under the Prichard regimen and other 30 were enrolled under Dhaka regimen.

History:

A detailed history regarding age, parity, gestational age, number of convulsions, duration of symptoms of pregnancy induced Hypertension, H/o imminent symptoms were taken from close relations and also from the patient if she is conscious (or) taken retrospectively from her. Any past history of hypertension (or) renal disease (or) Eclampsia in previous pregnancy was elicited.

Clinical Examination:

A through general examination and obstetric examination was made.

On general examination, conscious level, degree of edema, anaemia, blood pressure pulse rate, temperature, respiratory rate, cardiovascular

system, Respiratory system, fundus examination were done, Blood and urine were sent for all investigations related to eclampsia like Renal functions test, Liver functions, haematological investigations were carried out in all patients.

A life line was established and the Regimen was started. Hourly urine output was measured by an indwelling catheter. Half hourly pulse, temperature and respiratory rate, two hourly blood pressure were taken. Serum magnesium levels measured.

ANTI CONVULSANT LINE OF MANAGEMENT

1) DHAKA REGIMEN OF MAGNESIUM SULPHATE REGIMEN:

Loading Dose:

- 4gm of magnesium sulphate given intravenously slowly over 15minutes.
- 3gm given intramuscularly in each buttock.

Maintenance Dose:

- 2.5gm every 4 hours given intramuscularly in alternate buttocks, until 24hrs after administration of the first dose.
- Monitored with urine output kneejerks, and respiratory rate.

2) PRITCHARD REGIMEN OF MAGNESIUM SULPHATE REGIMEN

4gm of Magnesium sulphate ($Mgso_4$, $7H_2o$, USP) as a 20% solution intravenously at a rate not to exceed 1gm/min. Follow promptly with 10gm of 50% Magnesium sulphate solutions 5gm deep IM in each buttock.

5gm of 50% solution of magnesium sulphate every 4hours thereafter for 24 hours after delivery provided,

a) Patellar reflex is Present.
b) Respiratory Rate > 16/min
c) Urine output the previous 4hr exceeded 100ml.

Anti Hypertensive Line of Management:

Control of Hypertension achieved by T. Alphamethyl Dopa 250mg thrice daily and T. Nifidipine 10mg twice daily.

Obstetric Management:

After stabilizing the patient, a detailed obstetric examination was done. Mode of termination was planned according to the gestational age, viability of the fetus, and the cervical scoring.

Patients were induced with prostaglandin E_2 gel and accelerated with Oxytocin infusion.

Cesarean section was done for obstetric indications (or) for failed inductions.

After delivery the patient was observed carefully for 48 – 72 hours in the labour ward and post operative ward and followed up till the discharge of the patient.

Neonatal outcome was recorded in terms of Apgar scoring and birth weight. Neonates also followed up till the discharge of the mother.

Outcome Measures:

Primary outcome measures are recurrence of fits after starting the treatment in both the regimens. Perinatal mortality and maternal morbidity were compared in both groups.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

The study was done to compare the effectiveness of 2 regimens of magnesium sulphate Pritchard & Dhaka regimens. All woman with antepartum eclampsia were eligible and follow up was until discharge from hospital.

A. CHARACTERISTICS OF THE CASES STUDIED

Table : 1 Age

Age Group	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No	%	No	%
< 20	2	6.7	4	13.3
20 – 24	18	60	18	60
25 – 29	8	26.7	6	20
30 & above	2	6.7	2	6.7
Total	30	100	30	100
Mean	23.1		22.8	
S.D	3.6		3.6	
P	0.7711			

Age of Women in the two groups does not differ significantly.

In this study 6 cases (10%) were below 20years, 36 cases (60%) were between 20-24yrs, 14 cases (23%) were 25-29years and 4 cases (7%) were 30 years and above.

The mean age for Pritchard regimen is 22.8years and Dhaka regimen was 23.1yrs and the P value is 0.7711 which is insignificant.

Table : 2 Booking Status

Booking Status	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No	**%**	**No**	**%**
Booked	10	33.3	11	36.7
Un booked	20	66.7	19	63.3
P	0.7883 (Not Significant)			

In Pritchard regimen the booked cases were 36.7% in Dhaka regimen the booked cases were 33.3%.

The unbooked cases of Pritchard regimen was 63.3% and Dhaka Regimen was 66.7%

The p value of 0.7883 was insignificant.

Table : 3 Parity

Parity	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No	**%**	**No**	**%**
Primi	20	66.7	12	40
Multi	10	33.3	18	60
P	0.07			

Parity in the two groups does not differ significantly.

In our study 32 cases were primis. In Dhaka regimen 66.7% were primis. In CET trial group (1995) – 64% were primis. According to mudhaliar over 75% were primis. The p value being 0.07, the parity in two groups does not differ significantly.

Table : 4 Gestational Age

Gestational Age	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No	**%**	**No**	**%**
< 24 weeks	4	13.3	4	13.3
25 – 28	3	10	5	16.7
29 – 32	9	30	9	30
33 – 36	9	30	7	23.3
> 36	5	16.7	5	16.7
Mean	31.87		31.13	
S.D	4.63		4.89	
P	0.56			

In our study 8 cases (13.3%) were below 24weeks size (13.35%) 8 cases were 25-28 weeks. 18 cases (30%) were 29-32 weeks, 16 cases (26.6%) were between 33-36weeks. 10 cases (16.7%) were > 36 weeks size.

The mean gestational age of Pritchard regimen was 31.13 weeks. The mean gestational age for Dhaka Regimen was 31.87 weeks. The p value is 0.56, which is insignificant.

In a study by Katz and colleagues 2000 the mean gestational age during seizures was 34.2 weeks.

Table : 5 Level of Consciousness

Level of Consciousness	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No	%	No	%
Conscious	9	30	11	36.7
Semi conscious	21	70	19	63.3
P	0.7842			

Level of consciousness of the mothers in the two groups does not differ significantly.

The conscious patients on Pritchard Regimen 11 cases (36.7%) Dhaka Regimen 9 cases (30%). The semiconscious patients in Pritchard Regimen 19 cases (63.3%) Dhaka Regimen 21 cases (70%).

The p value being 0.7842 the level of consciousness of the mothers in both groups does not differ significantly.

Table : 6 Number of fits before admission

No of fits before admission	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)		P
	No.	%	No.	%	
1-2	14	46.7	20	66.7	0.5165
3-5	13	43.3	9	30	
6-8	2	6.7	1	3.3	
>9	1	3.3	0	0	
Mean	2.93		2.4		
S.D.	2.46		1.35		

Total number of fits before admission ranged between 1-9 fits giving a mean value of 2.93 at Dhaka regimen group and 2.4 in Pritchard Regimen Group.

The p value being 0.5165 the number of fits in both regimens carries insignificant role.

Table 7 : Recurrence of convulsions after starting the Regimen

Convulsions after starting the regimen	Group A (Dhaka regimen)		Group B (Pritchard regimen)	
	No.	%	No.	%
Nil	-	-	-	-
1	3	10	4	13.3
>1	-	-	-	-
P	0.5 (Not significant)			

In Group A 10% had one convulsion and Group B 13.3% had one convulsion and they needed a repeat dose of magnesium sulphate and the fits were controlled – none had more than one convulsion.

Table : 8 Hypertension

B.P	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No.	**%**	**No.**	**%**
SBP				
120-140	3	10	3	10
140-160	15	50	17	56
>160	12	40	10	34
DBP				
80-90	3	10	2	6.7
100-110	22	73	18	60
>110	5	17	10	33.3
P	0.7407 (Not Significant)			

- Majority of the cases have systolic blood pressure of 140-160 mm Hg - Dhaka Regimen group (50%) and in Pritchard Regimen group 56%.

- Majority of the cases have diastolic blood pressure of 100-110 mm Hg - Dhaka Regimen group (73%) and in Pritchard Regimen group 60%.

The p value being 0.7407 the diastolic BP doesn't make much difference in both regimens.

Table : 9 Serum magnesium mg/dl

Serum Magnesium	Group A (Dhaka Regimen)	Group B (Prichard Regimen)
Mean	4.69	5.01
S.D	0.3	0.4
P	0.0023	

- The mean serum magnesium level of Pritchard regimen was 5.01mg/dl
- The Dhaka regimen was 4.69mg/dl
- Both were within the therapeutic levels without going for toxicity.

Table 10 : Mode of Induction

Mode of induction	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No	**%**	**No**	**%**
Syntocinon	7	23.2	5	16.7
Gel	23	76.7	25	83.3
P	0.7469			

★ In 30 cases of Pritchard Regimen 5 cases induced with Syntocinon and 25 cases with Prostaglandin E_2 gel.

★ In 30 cases of Dhaka Regimen 7 cases were induced with Syntocinon and 23 cases with Prostaglandin E_2 gel.

The p value being 0.7469 which is insignificant.

B. OUTCOME IN THE TWO GROUPS

Table : 11 Mode of delivery

Mode of delivery	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No	%	No	%
Vaginal	27	90	28	93.3
LN delivery	23	76.7	24	80
Outlet forceps	3	10	3	10
Assisted Breech Delivery	1	3.3	1	3.3
C.S	3	10	2	6.7
P	0.5 (Not Significant)			

Out of 30 cases of Pritchard regimen 24 cases delivered by labour natural 3 cases by forceps 1 case as assisted breech delivery and 2 cases by LSCS.

Out of 30 cases of Dhaka Regimen 23 cases delivered by labour natural 1 cases as assisted breech delivery of 3 cases by forceps and 3 cases by LSCS.

- ★ Out of 5 cases delivered by LSCS.
- ★ 2 cases were due to failed induction.
- ★ 2cases were due to fetal distress.
- ★ 1 case due to impending renal failure.

Table : 12 Admission to delivery interval

Admission to delivery interval	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No.	**%**	**No.**	**%**
< 6 hours	5	17.9	7	23.3
6.1 – 12 hours	15	53.6	11	36.7
12.1 – 18 hours	6	21.4	10	33.3
> 18 hours	2	7.1	2	6.7
Mean	10.97 hours		11.81 hours	
S.D	4.1		5.37	
P	0.5926			

The mean duration of Admission – Delivery interval for Pritchard Regimen was 11.81hours and for Dhaka Regimen was 10.97 hours. In both Regimens only 2 cases in each delivered after 18 hours.

Table : 13 Condition of mother after delivery

Condition of mother	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No.	%	No.	%
Alive	30	100	30	100
Dead	-	-	-	-

In both Regimens there is no maternal mortality and they followed up in labour ward for 48-72 hours.

Table : 14 Condition of child after delivery

Condition of Child	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No.	%	No.	%
Alive	18	62.1	19	63.3
Still born	4	13.8	5	16.7
Neonatal death	7	24.1	6	20.0
P	0.7923 (Not Significant)			

Perinatal Outcome:-

- ★ Born alive in Pritchard regimen was 19 cases (63.3 %) Dhaka Regimen 18 cases (62.1%).
- ★ Still born in Pritchard Regimen was 5 cases (16.7%)
  In Dhaka Regimen 4 cases (13.8%)
- ★ The neonatal death with Pritchard regimen 6 cases (20%). In Dhaka Regimen 7 cases (24.1%)

The p value being 0.7923 the condition of the child doesn't differ significantly in both the regimens.

Table 15 : Birth weight of babies in the two groups

Birth weight (in kg)	Group A (Dhaka Regimen)		Group B (Pritchard Regimen)	
	No.	%	No.	%
< 1	3	10	3	10
1-1.5	7	23.3	7	23.3
1.6-2.5	15	50	14	46.7
>2.5	5	16.7	6	20
Mean	1.84		1.81	
S.D.	0.61		0.58	
P	0.801 (Not significant)			

There is no significant difference in the birth weight of the children in the two groups.

Perinatal mortality in relation to the birth weight of babies

Birth weight (in kg)	Cases	Perinatal Mortality	%
< 1	6	5	83.3
1-1.5	14	8	57.1
1.6-2.5	29	7	24.1
>2.5	11	2	18.2

As birth weight increases, perinatal mortality decreases.

Table 16 : Perinatal mortality Rate

	Total Cases	Perinatal deaths	
		No.	%
Group A	30	11	36.7
Group B	30	11	36.7

The perinatal mortality rate does not differ in both the regimens.

Maternal Morbidity

Dhaka Regimen	No.	%
Wound infection	1	3.35
Urinary tract infection	2	6.65
Puerperal psychosis	1	3.35
Puerperal sepsis	2	6.65

Pritchard Regimen	No.	%
Hemiplegia improved with Physiotherapy	1	3.35
Urinary tract infection	2	6.65
Puerperal sepsis	1	3.35
Wound infection	2	6.65

DISCUSSION

DISCUSSION

Prevention of further fits in eclampsia is associated with a reduction in adverse outcomes.[62] Magnesium is an ideal drug, with rapid onset of action, a non sedative effect on mother and baby, a fairly wide safety margin and a readily available antidote in the form of calcium gluconate.[63] The Collaborative Trial provided vital evidence that magnesium reduces the risk of recurrent seizures compared to other standard agents diazepam and phenytoin. Further more use of magnesium sulphate does not appear to be associated with detrimental effects on the neonate.[64]

Evidence from computed tomography and magnetic resonance angiographic studies implicating cerebral vasospasm and ischemia in the genesis of eclampsia.[65] Magnesium seems to reverse and ameliorate the effects of cerebral ischemia.[66]

There may also be a moderate inhibitory effects on cortical discharge. [67] With magnesium antagonizing the excitatory glutamate N-methyl aspartate receptor.[68]

Falling serum calcium levels following administration of Intra venous magnesium sulphate inhibit acetyl choline release at motor end plate. It depends upon serum calcium level.[69]

In our study we used total of 10gm $mgso_4$ as a loading does and 2.5gm 4 hourly which is just over half the dose used by Pritchard and an Collaborative Eclampsia Trial.

Age Distribution

A study in N.W.M. Hospital, Bombay in 1989 reveals that 40.5% were under 20 years, 56.8% were between 21-29 and 2.7% above 30 years. Lolkand et al in his study (1997) found that 40.7% were under 20 years. In a study by Katz VL and colleagues (2000) in the sacred heart medical center USA the mean age of eclampsia was 22 years.

In our study the mean age in Dhaka regimen is 23.1 years and mean age in Pritchard regimen is 22.8 years.

Parity

In the study of eclmapsia collaborative trial Group (1995) 64% were primis. In the study by N.W.M. Hospital, Bombay 1989 64.9% were primis. According to Mudaliar over 75% were primis. In a study by Lalkoand et al

(1997) 57.3% were primis. In our study in Dhaka regimen 66.7% were primis and in Pritchard regimen 40% were primis.

Gestational Age

In ECTG study 39.5% cases were less than 34 weeks and 25.5% cases were presented between 34-36 weeks and 33% cases were presented at term. In our study mean gestational age in Dhaka regimen was 31.87 weeks and in Pritchard regimen 31.13 weeks.

Diastolic Blood Pressure

In ECTG study 53% had a diastolic blood pressure above or equal to 110 mm Hg. In our study majority of cases were Diastolic blood pressure between 100-110 mm Hg and in Dhaka regimen 73% and in Pritchard regimen 60%.

Recurrence rate of convulsions

The recurrence rate of convulsions after starting the regimen in Dommisse (1990) – 0%, ECTG (1995) – 5.7%, PGI Chandigarh – 8.1%. In our study 10% in dhaka regimen and 13.3% in Pritchard regimen.

Mode of induction

Alexander and colleagues (1999) reviewed 278 singleton liveborn infants weighing 750-1500 gms delivered of woman with severe pre

eclampsia in Parkland hospital. 50% were induced and 50% underwent caesarean delivery without labor. Induction was not successful in 35% of women of induced group. Similar results were reported by Nassar and colleagues (1918).

In our study in Dhaka regimen 23.2 cases induced with syntocinon and 76.7% induced with prostaglandin E_2 gel. Among them 90% successfully delivered vaginally. 10% underwent caesarean section.

In Pritchard regimen 16.7% induced with syntocinon and 83.3% with prostaglandin E_2 gel.Among them 93.3% delivered vaginally and 6.7% by caesarean section due to failed induction and medical causes.

Perinatal Mortality

Perinatal deaths in Eclampsia Trial Collaborative Group (1995) with Magnesium sulphate was 25% with Diazepam 22% with Phenytoin 31%. In our study both Dhaka and Pritchard Regimens the perinatal death was 36.7%.

Maternal Mortality

The maternal mortality between 1991-1997 approximately 6% in US were related to eclampsia. (Berg & coworkers 2003). The maternal mortality with ECTG study 1995 5.2%, Eclampsia Trial Collaborative Group 1995 with

magnesium sulphate was 3.8%. In our study both the Dhaka regimen and Pritchard regimen no maternal death occurred.

No patient developed toxicity with low dose Dhaka Regimen. The earliest sign of toxicity would be loss of tendon reflexes which usually occur when serum levels of 10mg/dl are reached.

The range of serum magnesium concentration in Dhaka Regimen was 4.69mg%. This lies within Pritchard's therapeutic range.

In our study 76% patients regained consciousness within 6-12 hours.

Recent evidence has suggested that in-utero exposure to magnesium may be associated with higher 1 minute Apgar score and a lower prevalence of cerebral palsy.

SUMMARY

SUMMARY

No. of Cases	Dhaka Regimen 30	Pritchard Regimen 30
Recurrence of fits **No. of cases** (%)	 3 10%	 4 13.3 %
Serum Magnesium Level Mean Level	 4.69 mg %	 5.01 mg %
Mode of Delivery Vaginal LSCS	 27 (90%) 3 (10%)	 28 (93.3%) 2 (6.7%)
Perinatal Outcome Live Born Still Born Neonatal death PNMR	 18 (62.1%) 4 (13.8%) 7 (24.1%) 36.7%	 19 (63.3 %) 5 (16.7%) 6 (20%) 36.7%
Maternal Morbidity	7 (23.3%)	6 (20%)

- In this study, 30 eclamptic cases were treated with Pritchard regimen and compared with another 30 cases which were treated with Dhaka regimen.
- Recurrence of fits after starting the regimen was lower in both the magnesium sulphate regimen groups.
- Only 4 cases (13.3%) in the Pritchard group had recurrence of fits, whereas, 3 cases (10%) in the Dhaka group had recurrence of fits.
- 80 % cases in both groups delivered within 18 hours of admission.

- 93.3% (28 cases) in the Pritchard group and 90 % (27 cases) in the Dhaka group delivered vaginally.
- The mean serum magnesium level of Pritchard regimen is 5.01 mg% and Dhaka regimen is 4.69 mg%.
- 2 cases (6.7%) in the Pritchard group and 3 cases (10%) in the Dhaka group are delivered by LSCS.
- 19 babies (63.3%) in the Pritchard group and 18 babies (62.1%) in the Dhaka group were born alive.
- 5 babies (16.7%) in the Pritchard group and 4 babies (13.8%) in the Dhaka group were still born.
- Maternal morbidity was 20% in Pritchard regimen and 23.3 % in Dhaka regimen.
- The perinatal mortality rate doesn’t differ in both the groups.

CONCLUSION

CONCLUSION

Magnesium sulphate is the anti convulsants drug of choice in woman with eclampsia.

The low dose Dhaka Regimen used for smaller woman appears to control and prevent convulsions effectively.

Our study of Dhaka Regimen showed that serum levels remain below the toxic levels.

The present study provides further strong support for the routine use of magnesium sulphate for eclampsia convulsions.

As long as there is adequate urinary output clinical monitoring appears to be sufficient.

There is no difference in maternal mortality, perinatal mortality, maternal morbidity and cesarean section rates among the both magnesium sulphate regimens.

Like the Pritchard regimen the admission to delivery interval was lower in Dhaka regimen.

The study clearly shows that Dhaka regimen is almost equivalent to Pritchard regimen, on control of convulsions on eclampsia cases.

Proper antenatal care, improved socio economic status and intensive management will largely reduce the incidence of eclampsia which is an important cause of maternal morbidity and perinatal mortality.

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PROFORMA

PROFORMA

NAME	:	BOOKED	:	ABORTION	:	LMP	:
AGE	:	UN BOOKED	:	URBAN	:	EDD	:
IPNO.	:	GRAVIDA	:	RURAL	:		
UNIT	:	PARA	:	SOCIO ECONOMIC STATUS:			

DATE AND TIME OF ADMISSION :

DATE AND TIME OF DELIVERY :

DATE OF DISCHARGE :

COMPLAINTS :

PAST HISTROY :

MEDICAL

SURGICAL

MENSTRUAL HISTORY :

MARITAL HISTORY :

PERSONAL HISTORY :

FAMILY HISTORY :

PREVIOUS OBSTETRIC HISTROY :

HISTROY OF PRESENT PREGNANCY :

GENERAL EXAMINATION :

OBSTETRIC EXAMINA TIQN :

MODE OF DELIVERY :

VAGINAL : NORMAL

INSTRUMENTAL

LSCS : INDICATION FOR LSCS

MATERNAL COMPLICATIONS :

FOETAL OUTCOME : SEX

WEIGHT

APGAR

LIVE BORN / STILLBORN / NEONATAL DEATH

INVESTIGATIONS

URINE

- ALBUMIN
- SUGAR
- DEPOSITS
- CULTURE AND SENSITIVITY

Hb%

BLOOD

- GROUPING AND TYPING
- UREA
- SUGAR
- CREATININE
- URIC ACID

SERUM MAGNESIUM

PLATE LET COUNT

BLEEDING TIME

CLOTTING TIME

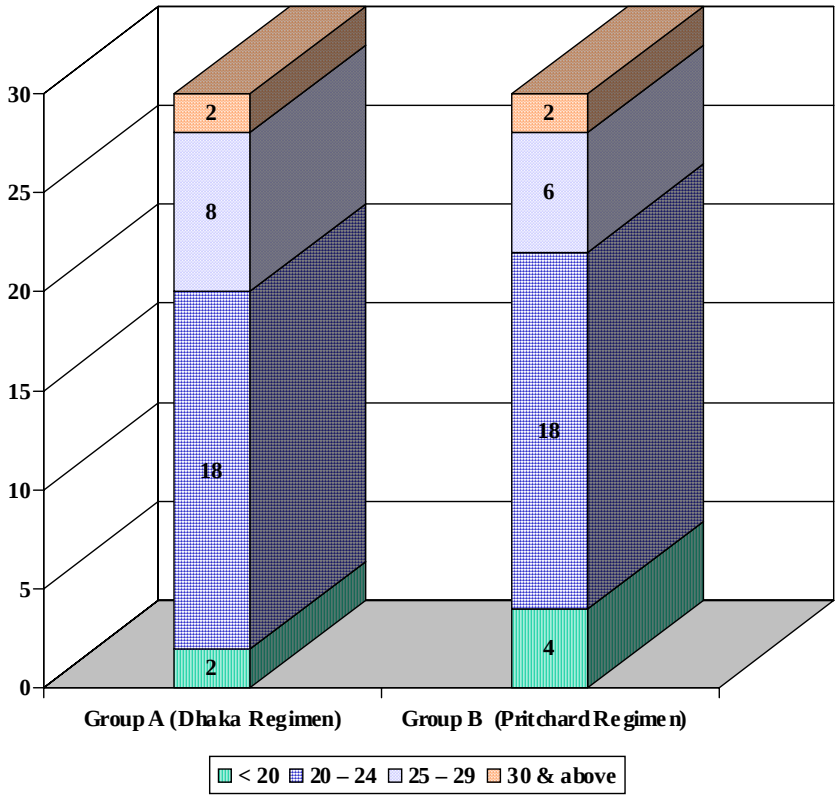
PLASMA FIBRINOGEN

LIVER FUNCTION TESTS

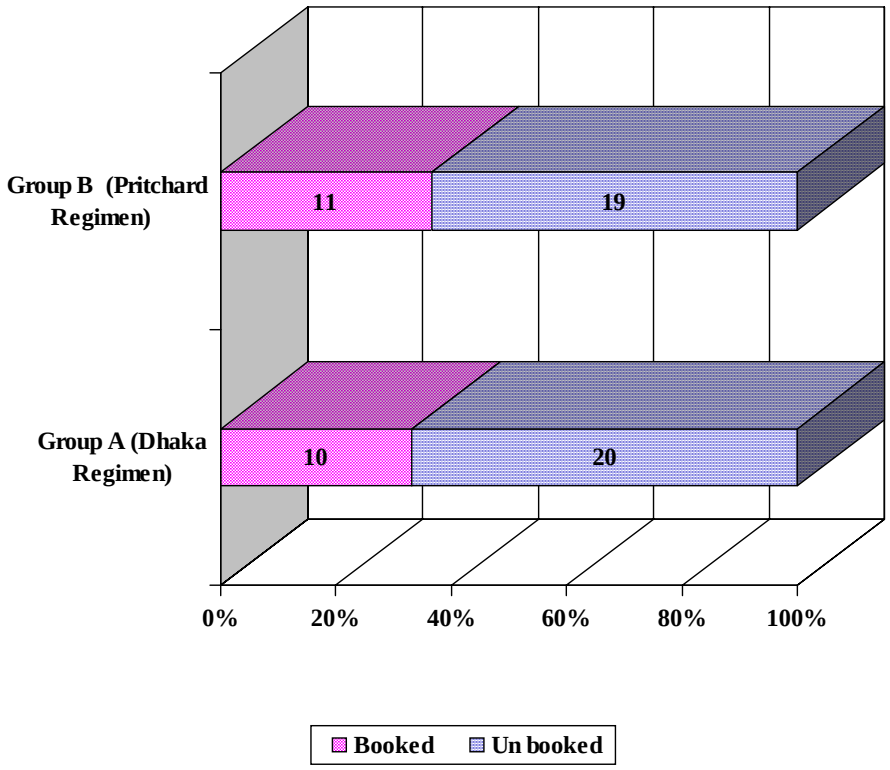
FUNDOSCOPY

USG

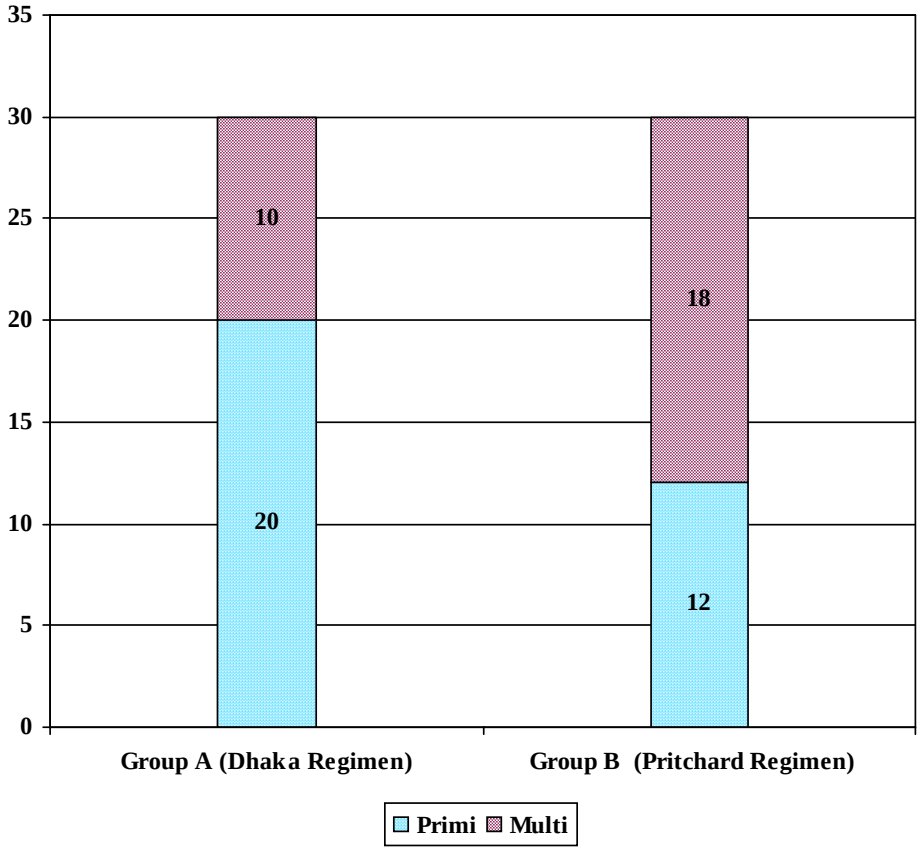
Age


30
25
20
15
10
5
0
2
8
18
2
2
6
18
4
Group A (Dhaka Regimen)
Group B (Pritchard Regimen)
< 20
20 – 24
25 – 29
30 & above

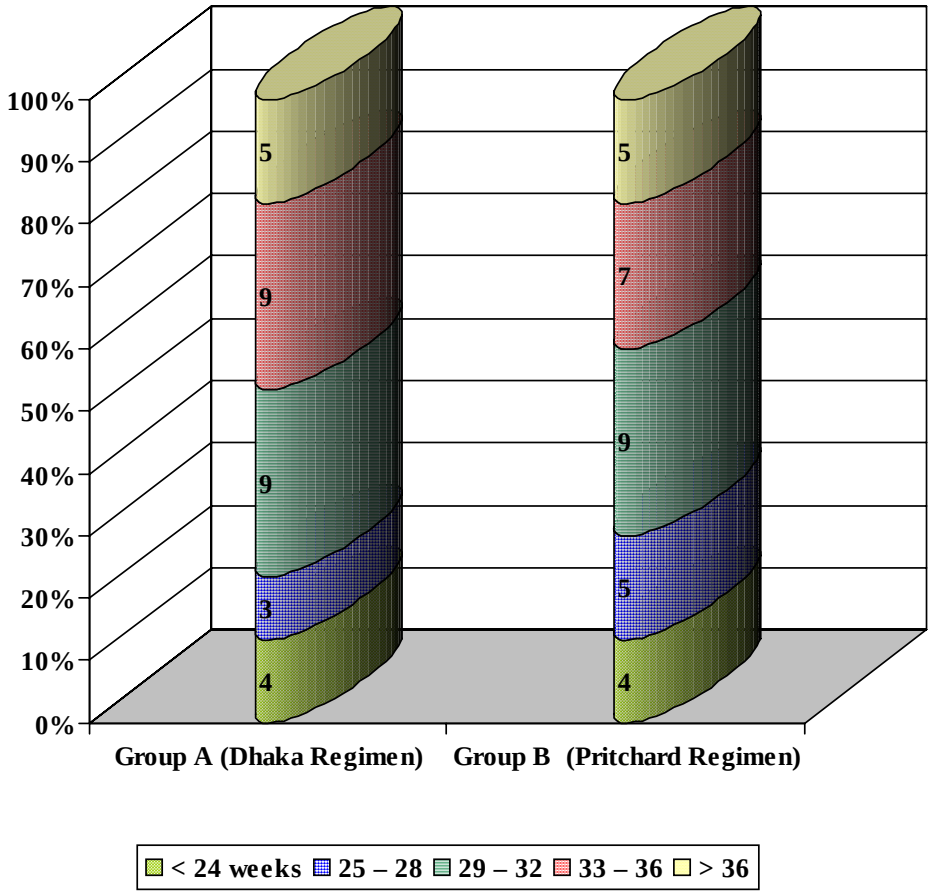
Booking Status



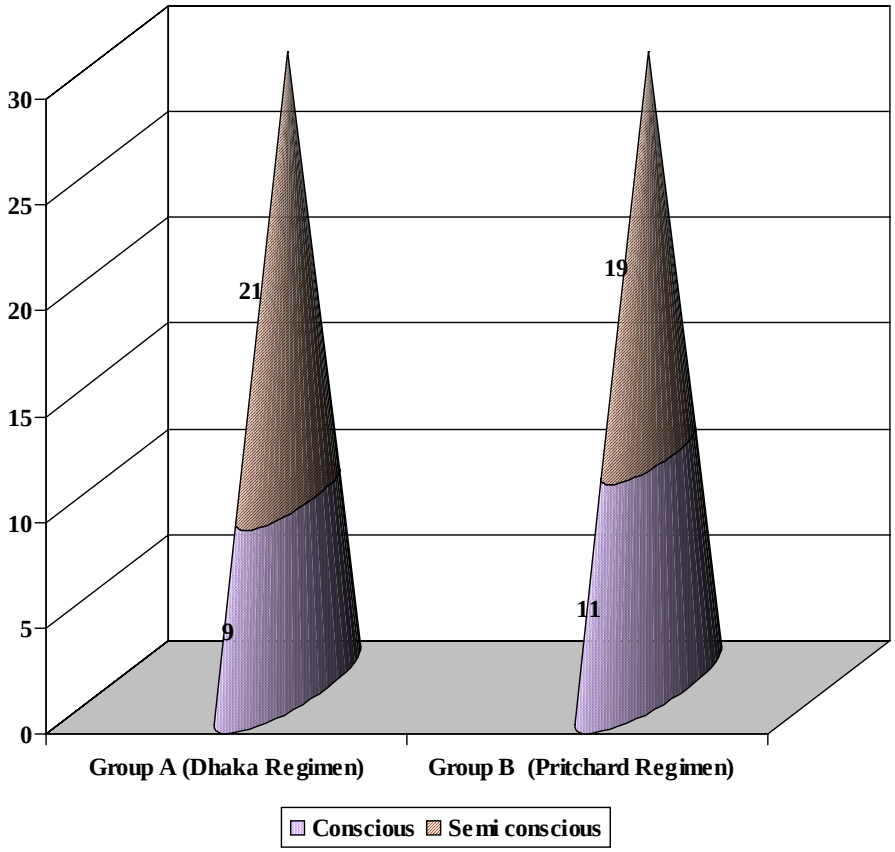
Parity


35
30
25
20
15
10
5
0
10
18
20
12
Group A (Dhaka Regimen)
Group B (Pritchard Regimen)
Primi
Multi

Gestational Age

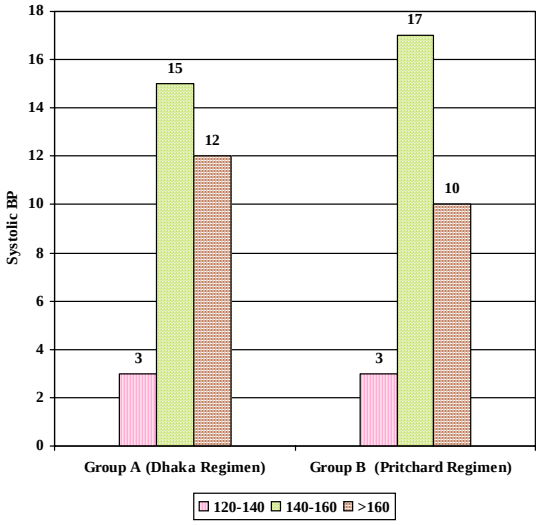


Level of Consciousness

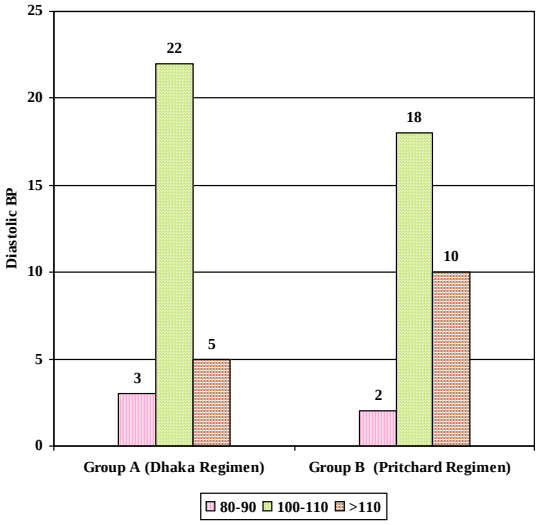


Hypertension

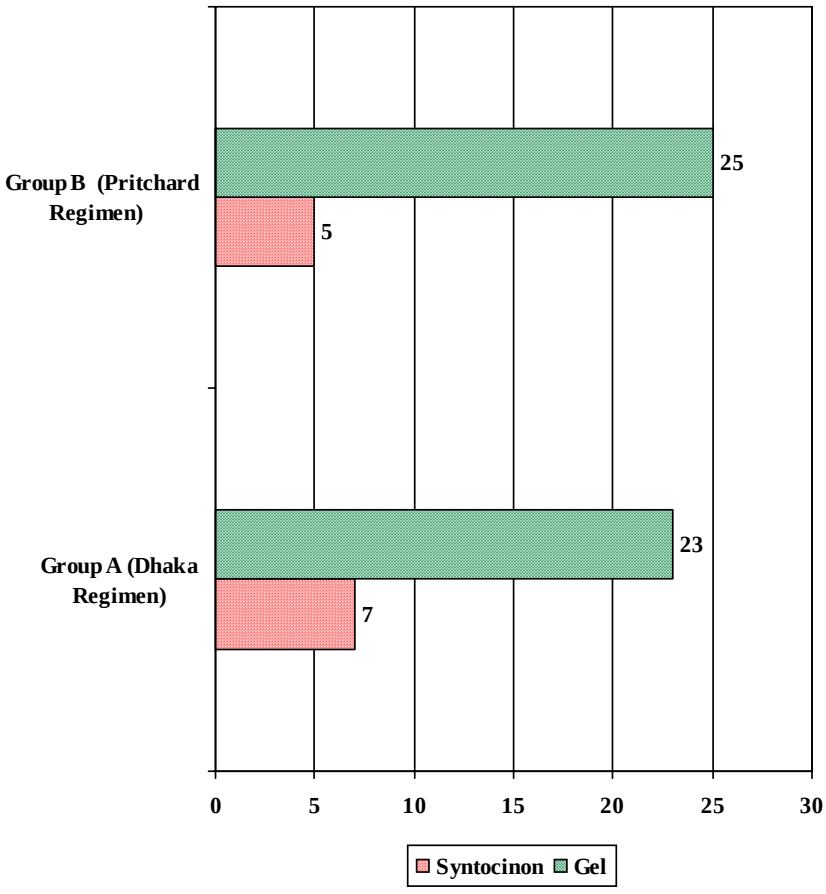
Systolic BP



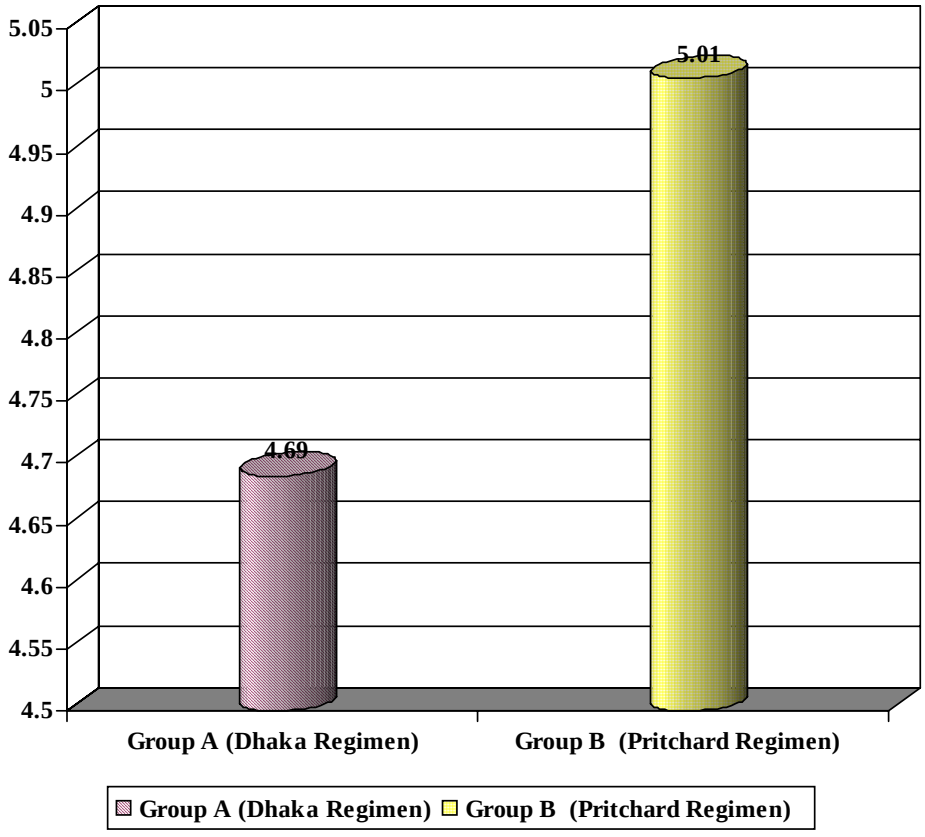
Diastolic BP



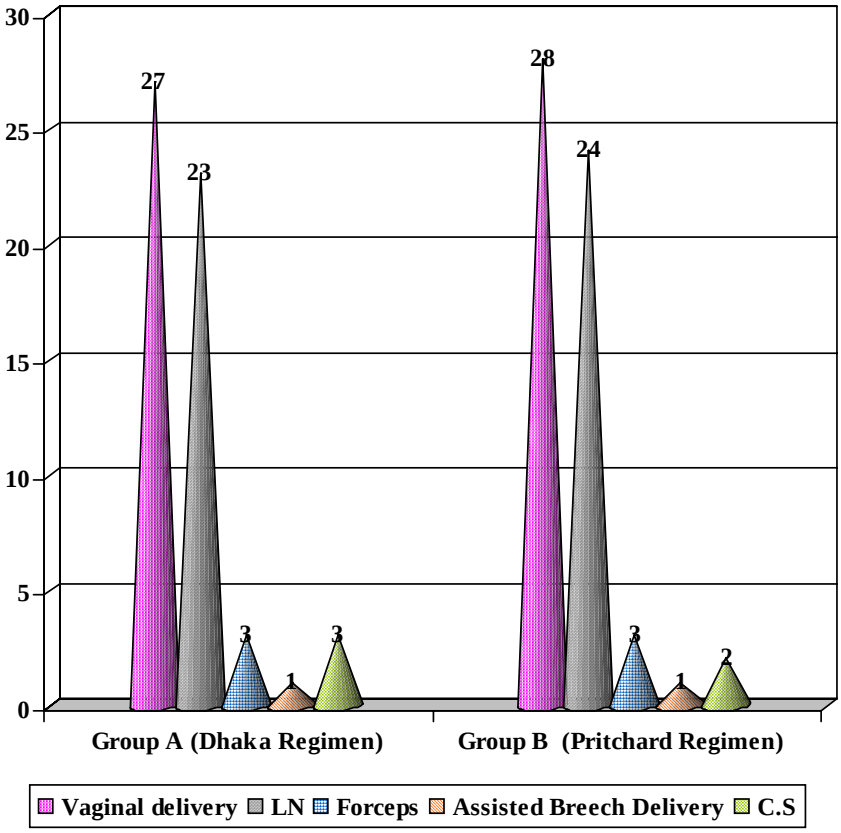
Mode of Induction



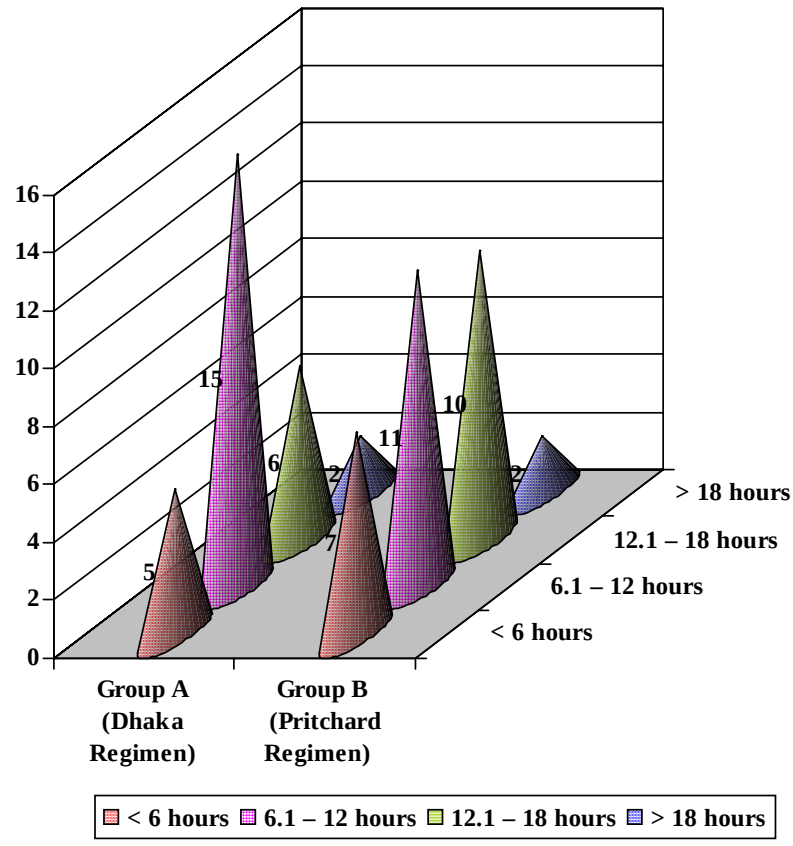
Serum Magnesium (Mg/dl.)



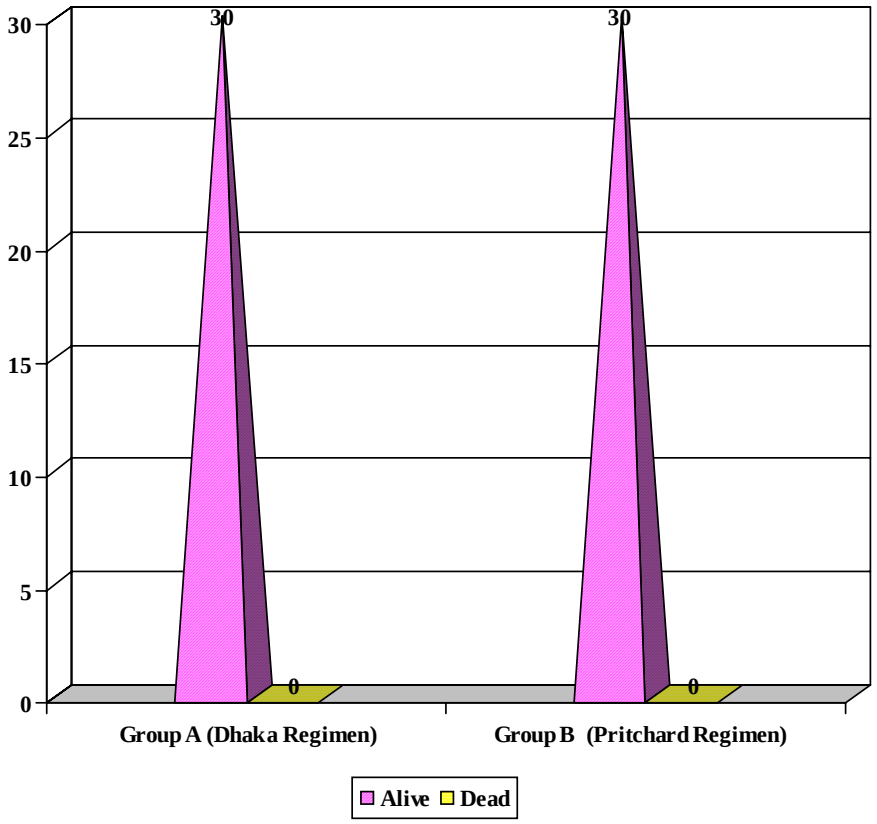
Mode of Delivery



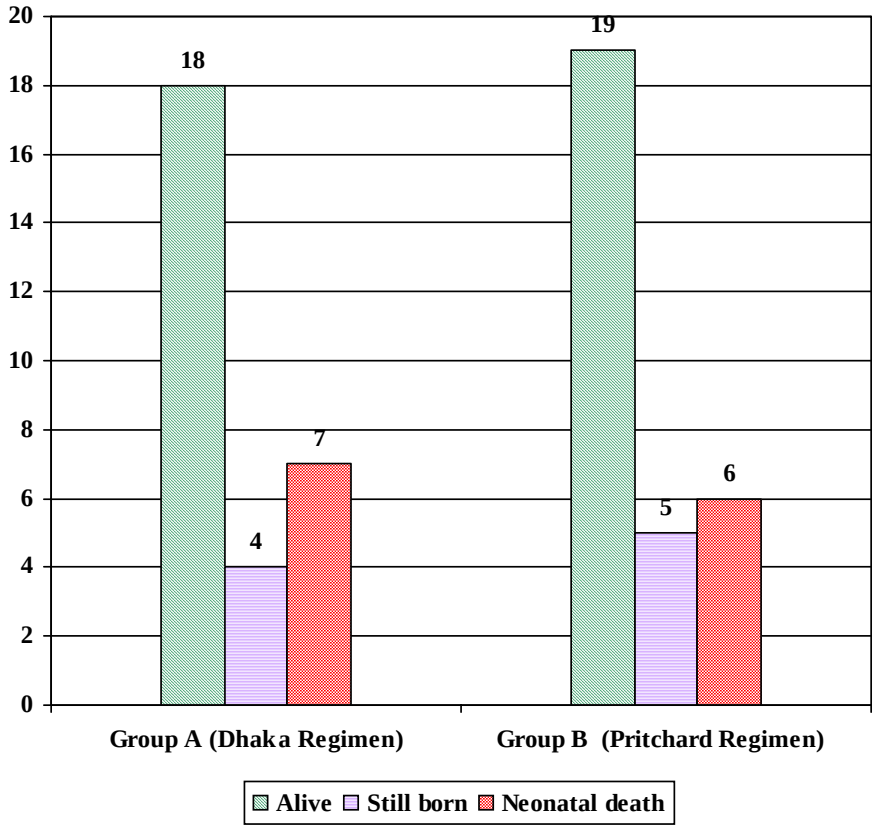
Admission to Delivery Interval



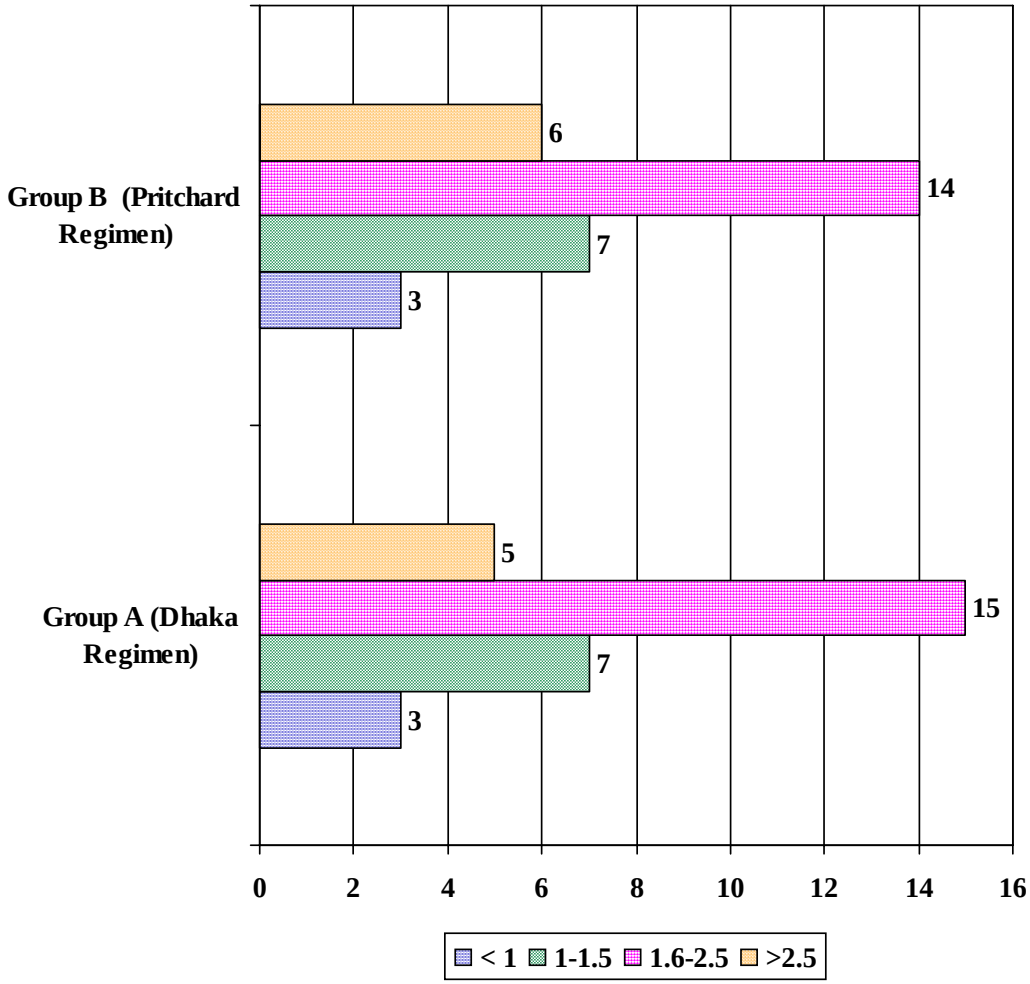
Condition of mother after delivery



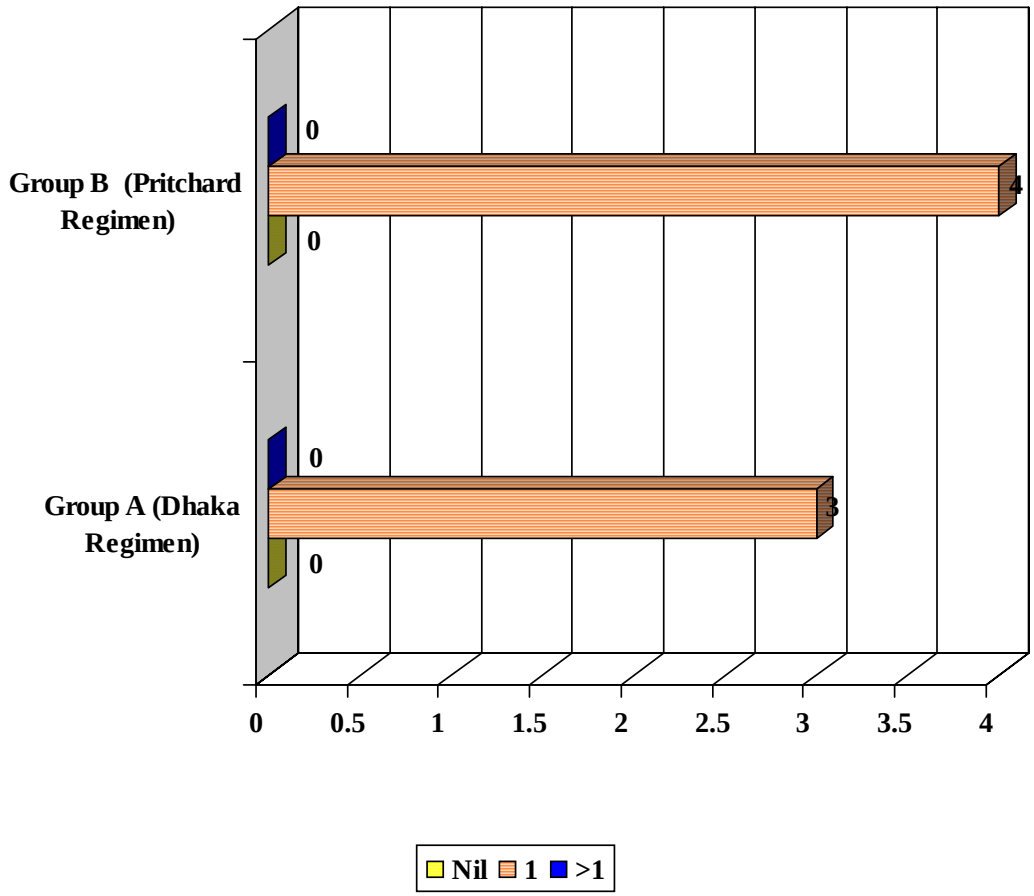
Condition of child after delivery



Birth Weight of Babies in the two groups



Recurrence of Convulsions after starting the Regimen



MASTER CHART

S.No.	Name	Group	Age yrs	Booked Unbooked	parity	Obst. Code	GA wks	Level of cons. At adm.	No. of fits before adm.	HT	BP SYS	DIA	Sr. Mg. mg/ dl	MOI	MOD	Admn. Deliver interval hrs	Condition after del mother	B. Weight kg	Baby	No.of fits after deli.
1	Mariammal	A	25	2	MULTI	G2P1L1	32	2	10	2	150	100	4.4	1	1	6	1	1.7	1	0
2	Mariammal	A	23	1	MULTI	G2A1	30	2	2	1	140	100	4.2	2	1	9	1	0.9	2	0
3	Sundarammal	A	33	1	MULTI	G3P2L2	34	2	2	2	150	100	4.6	2	1	7	1	1.75	1	0
4	Kavitha	A	25	1	MULTI	G2P1L1	24	1	3	2	150	110	4.2	1	1	13	1	1.1	3	1
5	Ponselvi	A	20	2	Primi	Primi	36	1	3	1	140	100	4.8	2	1	8	1	2	1	0
6	Mekala	A	25	1	Primi	Primi	36	2	3	2	200	120	5	1	1	10	1	2.1	1	0
7	Pullani	A	25	1	Primi	Primi	24	2	3	2	170	110	4.8	2	2	19	1	1.25	2	0
8	Noorjahan	A	21	1	Primi	Primi	34	1	1	2	150	120	4.6	2	2	16	1	2.7	3	0
9	Ponnalagu	A	20	2	MULTI	G2P1L1	24	2	3	2	200	120	4.2	1	1	6	1	0.9	2	0
10	Mareeswari	A	20	2	Primi	Primi	28	1	1	2	190	110	5	2	1	9	1	1.3	3	1
11	Selvi	A	24	2	Primi	Primi	34	2	1	2	220	140	4.8	1	1	3	1	2.3	1	0
12	Indira	A	19	2	MULTI	G2A1	28	2	3	2	160	100	4.6	2	1	12	1	1.5	1	0
13	Pitchaiammal	A	20	2	Primi	Primi	38	2	2	2	180	120	4.4	1	1	4	1	32	1	0
14	Rojabans	A	22	2	Primi	Primi	30	2	4	2	150	100	4.8	2	1	12	1	1.2	2	0
15	Selvi	A	26	2	Primi	Primi	38	1	3	2	150	100	5.2	2	1	19	1	2	1	0
16	Usha	A	22	2	Primi	Primi	36	1	1	2	150	100	4.6	2	1	12	1	2.8	1	0
17	Hema	A	21	1	Primi	Primi	30	2	2	1	120	90	4.2	2	1	15	1	1.5	1	0
18	Janasamariamm	A	21	2	MULTI	G2P1L1	38	2	1	2	190	110	5	2	1	12	1	2	1	0
19	Muniammal	A	21	2	MULTI	G2P1L1	32	2	1	2	180	110	4.6	1	1	15	1	2	1	0
20	Petchi	A	32	2	MULTI	G2P1L1	28	2	5	2	150	100	4.8	2	1	12	1	1.9	1	0
21	Kalaiselvi	A	25	2	Primi	Primi	30	2	4	2	160	110	5.2	2	1	11	1	2.4	1	0
22	Meenachi	A	22	2	MULTI	G2P1L1	38	1	1	1	140	100	4.4	2	2	8	1	2.8	1	0

S.No.	Name	Group	Age yrs	Booked Unbooked	parity	Obst. Code	GA wks	Level of cons. At adm.	No. of fits before adm.	HT	BP SYS	DIA	Sr. Mg. mg/ dl	MOI	MOD	Admn. Deliver interval hrs	Condition after del mother	B. Weight kg	Baby	No.of fits after deli.
23	Sabrisa Banu	A	28	2	Primi	Primi	30	2	4	2	160	110	4.8	2	1	10	1	1.1	3	0
24	Muthammal	A	20	2	Primi	Primi	34	2	2	2	180	110	5	2	1	11	1	2	1	1
25	Chitra	A	28	1	Primi	Primi	32	1	3	2	160	100	5.2	2	1	10	1	1.9	3	0
26	Rajalakshmi	A	24	1	Primi	Primi	36	2	1	1	130	80	4.6	2	1	18	1	2	3	0
27	Pitchaiammal	A	21	2	Primi	Primi	30	2	2	2	150	90	4.8	2	1	10	1	1.6	1	0
28	Murugeswari	A	20	1	Primi	Primi	38	2	3	2	160	110	4.6	2	1	12	1	2.6	3	0
29	Muthulaxmi	A	18	2	Primi	Primi	22	2	12	2	200	100	4.4	2	1	6	1	0.9	-	1
30	Latha	A	22	2	Primi	Primi	32	1	2	2	160	100	4.8	2	1	14	1	1.8	1	0
31	Murugasali	B	22	1	Primi	Primi	38	2	1	2	200	160	4.6	3	1	18	1	2.2	1	0
32	Selvi	B	24	1	Primi	Primi	22	1	2	2	150	110	4.8	2	1	9	1	0.9	2	0
33	Chitra	B	24	2	MULTI	G2P1L1	30	2	6	2	160	120	5.2	2	1	6	1	0.8	2	0
34	Thanalaxmi	B	22	2	Primi	Primi	38	2	2	2	190	150	4.8	1	1	10	1	2.6	1	1
35	Shanthi	B	20	1	Primi	Primi	24	2	1	1	130	100	5.2	2	1	16	1	1.7	3	0
36	Gurupriya	B	22	2	Primi	Primi	30	1	4	1	130	90	5	2	1	6	1	1.5	2	0
37	Meena	B	24	2	MULTI	G2P1L1	30	1	2	2	150	100	5.4	1	1	8	1	2	1	0
38	Nagalaxmi	B	18	2	MULTI	G2P1L1	34	2	3	2	160	110	5.2	1	1	8	1	1.8	1	1
39	Murugeswari	B	20	1	MULTI	G3A2	28	2	4	2	170	110	4.6	1	1	12	1	3.2	3	0
40	Karthigai Rani	B	24	1	MULTI	G2P1L1	38	2	1	2	200	150	4.8	2	1	12	1	1.6	1	0
41	Lakshmi	B	24	1	MULTI	G2P2L2	30	1	1	2	170	110	4.4	2	1	22	1	1.6	3	0
42	Rajeswari	B	20	2	Primi	Primi	28	2	2	1	140	100	4.8	2	1	16	1	1.1	1	0
43	Arulmozhi	B	23	1	Primi	Primi	36	2	2	2	160	110	4.4	2	2	6	1	2	1	0
44	Punithavathi	B	20	1	Primi	Primi	30	2	2	2	160	120	5.8	2	1	18	1	1.75	1	0
45	Muniammal	B	25	2	Primi	Primi	34	2	3	2	170	110	4.8	2	1	22	1	1.6	3	0
46	Shanthi	B	26	2	MULTI	G3P2L2	38	1	1	2	150	100	4.6	1	2	6	1	2.8	1	0

S.No.	Name	Group	Age yrs	Booked Unbooked	parity	Obst. Code	GA wks	Level of cons. At adm.	No. of fits before adm.	HT	BP SYS	DIA	Sr. Mg. mg/ dl	MOI	MOD	Admn. Deliver interval hrs	Condition after del mother	B. Weight kg	Baby	No.of fits after deli.
47	Selvapathi	B	30	2	MULTI	G3P1L1A1	34	1	2	2	150	100	5.2	2	1	10	1	2.5	1	0
48	Chironammal	B	18	1	Primi	Primi	34	2	4	2	170	130	4.8	2	1	6	1	1.8	1	0
49	Gandhi mathi	B	25	2	MULTI	G2P1L1	28	2	2	2	160	120	5.4	2	1	12	1	1.2	2	1
50	Alagurani	B	19	2	Primi	Primi	38	1	3	2	150	110	5.8	2	1	14	1	2	1	0
51	Radhalaxmi	B	25	2	MULTI	G4P2L1A1	32	2	2	1	110	70	4.8	2	1	18	1	1.5	1	0
52	Sundara pandiammal	B	20	1	Primi	Primi	34	1	1	2	150	110	5.6	2	1	7	1	1.9	1	0
53	Ramjan	B	20	2	Primi	Primi	20	2	2	2	160	120	5	-	1	0.3	1	2.1	1	0
54	Chandra	B	25	2	MULTI	G3P2L2	34	2	3	2	150	110	5.2	2	1	6	1	2.2	1	0
55	Manickam	B	22	2	Primi	Primi	26	1	5	2	170	120	5.4	2	1	14	1	1.2	3	0
56	Rajeswari	B	23	2	Primi	Primi	32	2	1	2	180	100	4.6	2	1	12	1	2.5	3	0
57	Muthurakku	B	19	2	Primi	Primi	30	1	1	1	140	100	5.6	2	1	15	1	1.5	1	0
58	Shanthi	B	35	2	Primi	Primi	24	1	2	1	140	110	4.8	2	1	16	1	900gms	2	1
59	Vijayalakshmi	B	25	2	MULTI	G2P1L1	28	2	5	2	180	120	5.2	2	1	18	1	1.5	1	1
60	Pitchaiammal	B	21	1	Primi	Primi	32	2	2	2	150	110	4.6	2	1	11	1	2.25	1	-

ABBREVIATIONS

GA	-	Gestational Age
BP Sys	-	Systolic blood pressure

Group

Group A	-	Dhaka Regimen
Group B	-	Pritchard Regimen

Booking Status

1	-	Booked
2	-	Unbooked

MOD	**-**	**Mode of Delivery**
1	-	Vaginal delivery
2	-	LSCS

Condition of mother after delivery

1	-	Alive
2	-	Still born
3	-	Neonatal death

Sr. Mg	-	Serum Magnesium
DIA	-	Diastolic blood pressure

Level of Consciousness

1	-	Conscious
2	-	Semi conscious

MOI	**-**	**Mode of Induction**
1	-	Syntocinon
2	-	Prostaglandin E2 gel

Condition of mother after delivery

1	-	Alive
2	-	Dead